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The oculomotor gap effect without a foveal fixation point

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Abstract

Turning off a fixation point prior to or coincident with the appearance of a visual target reduces the latency of saccades to that target. We investigated this 'gap effect' when subjects fixated a central point or the center of a square formed by four points that were 4, 2 or 1° eccentric from the square's center. The fixation anchor vanished 200 ms prior to the appearance of a saccadic target in a Gap condition, coincident with the target's appearance in a 0-Gap condition, or remained on in an Overlap condition. Saccadic reaction time was reduced in the Gap relative to 0-Gap condition irrespective of the type of fixation anchor. However, saccadic reaction time was not reduced in the 0-Gap relative to Overlap condition when the points forming the square had eccentricities of 2 or 4°. Results are interpreted in terms of a partial mediation of the gap effect by fixation cells in the rostral pole of the superior colliculus.

Keywords: Fixation cells; Gap effect; Saccadic reaction times; Superior colliculus

1. Introduction

Saccades to a suddenly presented visual target typically have latencies that range from 150 to 250 ms. However, if an observer's fixation point is turned off 200–300 ms before the target appears, saccadic latencies are significantly reduced (Saslow, 1967; Fischer & Ramsperger, 1984). This reduction in saccadic reaction time (SRT) has been termed the 'gap effect'. A smaller but significant latency reduction is found when the fixation point is turned off simultaneous with the target's appearance.

The mechanisms underlying these facilitatory effects have been a matter of controversy. An initial explanation based on the incidence of fixation microsaccades (Saslow, 1967) has been ruled out (Kingstone, Fendrich, Wessinger & Reuter-Lorenz, 1995), but anticipatory saccades (Kalesnykas & Hallett, 1987; Kingstone & Klein, 1993b), general response readiness (Ross & Ross, 1980; Kingstone & Klein, 1993a; Reuter-Lorenz, Oonk & Barnes, 1995), the early partial programming of motor (or specifically oculomotor) orienting responses (Kowler, 1990; Reuter-Lorenz, Hughes & Fendrich, 1991; Bekkering, Pratt & Abrams, 1996; Paré & Munoz, 1996), the 'disengagement of attention' (Fischer & Breitmeyer, 1987; Fischer & Weber, 1993) and a facilitated release from active fixation (Reuter-Lorenz, Hughes & Fendrich, 1991; Fendrich, Hughes & Reuter-Lorenz, 1991; Munoz & Wurtz, 1992; Kingstone & Klein, 1993a; Dorris & Munoz, 1995; Nozawa, Reuter-Lorenz & Hughes, 1995) have all been proposed as possible contributing factors.

Fixation related neurons in the rostral pole of the primate superior colliculus (SC) (Munoz & Wurtz, 1993a) provide a plausible neural substrate for explanations of the gap effect that are based on a facilitated release from active fixation. It has been hypothesized that these cells must be deactivated before a saccade can be initiated, and the offset of a fixation point facilitates this deactivation leading to reduced SRTs. (Munoz & Wurtz, 1992; Dorris & Munoz, 1995). Physiological data from the monkey provide support for this hypothesis: chemical deactivation of the colliculus rostral pole reduces saccadic latencies, while stimulation of this region serves to block saccades. (Munoz & Wurtz, 1993b). Moreover, changes in the discharge rate of rostral pole fixation neurons which occur when a fixation point is turned off reflect the time course of the gap effect (Dorris & Munoz, 1995).

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Normally, fixation entails holding a target steady on the fovea, which is the retinal region represented at the colliculus rostral pole. In the monkey, rostral pole fixation cells can be active during attempts to fixate an unmarked spatial location, but their activity is reduced relative to a condition in which a foveal stimulus is present (Munoz & Wurtz, 1993a; Dorris & Munoz, 1995). We therefore hypothesized that if the gap effect in humans is mediated by colliculus fixation cells, this effect would be reduced or eliminated by the use of an eccentric (non-foveal) fixation anchor. We tested this hypothesis by measuring the gap effect when subjects fixated the center of a square formed by four eccentric points.

2. Methods

Stimuli were presented on a Hewlett Packard 1311 X-Y display with a fast (P15) phosphor cathode ray tube (CRT) in a darkened room. Right eye movements were monitored with a Purkinje image eyetracker with a resolution of 1 arc min and stored for subsequent analysis. All testing was carried out monocularly with the right eye. Eye record sampling began 800 ms prior to the appearance of the saccadic target, and continued until the disappearance of that target 1.8 s later. Display refresh rates and eye position sampling were synchronous at 500 Hz. The presentation of stimuli and collection of records was controlled by a PC type computer interfaced to the eyetracker and Hewlett Packard display by Data Translation AD and DA converters. The luminance of the points making up the stimuli, measured by a Pritchard spot photometer with a close-up lens, was 20 cd m^{-2} against a dark (<1 cd m^{-2}) background.

Data was collected in blocks of 72 trials. In central fixation anchor blocks, subjects fixated a point at the center of the CRT display and initiated each trial with a button press. Each block contained 24 Gap trials, 24 0-Gap trials and 24 Overlap trials. During Gap trials, following a variable 900–1200 ms delay, the fixation point vanished for 200 ms. Following this gap, the saccadic target (a 0.5° 'x') appeared, horizontally 6° to the left or right of the central fixation position and remained on for 1 s. Subjects were instructed to saccade to the target as quickly as possible. On the 0-Gap trials, the disappearance of the fixation point was simultaneous with the targets appearance. In the Overlap trials, the fixation point remained on until the disappearance of the target. In an effort to control general warning effects, a small speaker mounted over the CRT sounded a 1200 Hz. tone during the final 200 ms prior to target's appearance on all three types of trials.

In eccentric fixation anchor blocks, stimuli and procedures were identical, save that subjects fixated at the center of a square formed by four points. In an initial set of experimental sessions, each point was 4° eccentric from square's center, as illustrated in Fig. 1. This center position corresponded to the location of the fixation point in central fixation anchor blocks.

Within each block, half of the target presentations for each condition were to the right, and half to the left, of the central fixation location. The order of trials was randomized within each block. In a first set of experimental sessions, four central and four eccentric fixation stimulus blocks were run in a counterbalanced order, yielding a maximum of 96 trials for each display condition. Data were collected from eight subjects, seven naive with respect to the purpose of the investigation. Prior to the start of data collection, subjects were run in one practice block with a central fixation anchor and one practice block with the eccentric fixation anchor. Three additional subjects failed to show any gap effect in either the central or eccentric fixation anchor practice blocks. Since these subjects could provide no information about how the gap effect would be influenced by the fixation condition, they were rejected from the experiment. All subjects were under age 25.

In subsequent experimental sessions, the points forming the fixation square were positioned 1 and 2° from the center of the square. Five of the subjects (four naive) run in the earlier sessions were tested with these stimuli. Four blocks were run with the 1 and 2° eccentric fixation anchors in a counterbalanced sequence.

3. Results

3.1. Fixation stability

To verify that subjects were not looking at a corner point of the peripheral fixation anchors, we assessed their fixation stability by measuring the arc min devia-

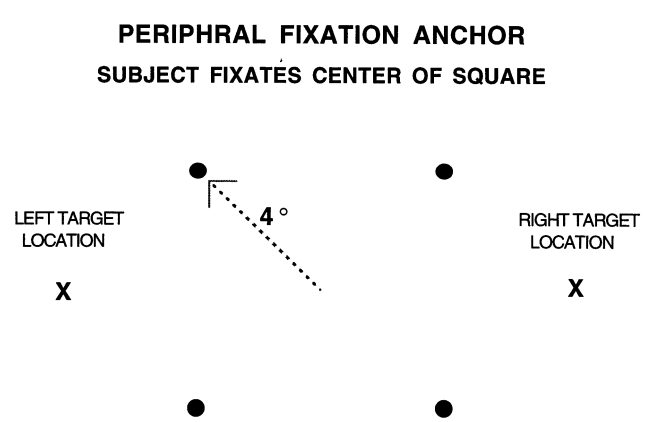

Fig. 1. The 4° eccentric fixation anchor. Subjects maintained fixation at the unmarked center of the square. In subsequent conditions the corner points of the square were 1 and 2° from the square's center.

tion of the eye from the center of the screen (defined as the subject's fixation position while looking at a central point during calibration of the eyetracker) at the time the target was presented. With the central fixation point the mean deviation was 16.1 min, with subjects looking within 1° of the fixation point on 99.7% of the trials. When the fixation anchor was a square with corner points 1° from its center, fixation stability was similar; the mean arc min deviation of gaze was 16.2 min, and subjects were looking within 1° of the square's center on more than 99.5% of the trials. With the 2° eccentric fixation anchor, fixation accuracy was only slightly worse with a mean error of 22 min and the gaze within 1° of center on 99.0% of the trials. With the 4° eccentric fixation anchor, the mean fixation error was 35 min and the subject's point of regard was within 1° of center on a mean of more than 94% of the trials. Fig. 2 plots the incidence of fixation deviations with both the central and eccentric fixation anchors for the subjects with the best and worst overall fixation stability. The ability of our subjects to achieve good fixation stability with the peripheral fixation anchors is in accord with the findings of previous investigators (Steinman, 1965; Rattle, 1968; Sanbury, Skavenski, Haddad & Steinman, 1973).

3.2. Saccadic amplitudes

Overall, the mean amplitude of saccades to the target measured at the endpoint of the primary saccade was 5.73°, just under the optimal value of 6°. Specific mean amplitudes for the 0, 1, 2 and 4° fixation anchor conditions were respectively, 5.81, 5.63, 5.7 and 5.76°. Collapsed across fixation condition, respective mean saccadic amplitudes for the Gap, 0-Gap and Overlap conditions were 5.65, 5.74 and 5.8°. A two-way ANOVA confirmed that neither the gap or fixation condition influenced saccadic amplitudes.

3.3. Saccadic latencies

Table 1 presents the mean SRTs for all subjects in all conditions, and the across subject means and standard deviations.

Saccadic latencies were analyzed using a two factor repeated measures ANOVA with fixation anchor type (central and eccentric) and gap condition (200 ms Gap, 0-Gap and Overlap) as factors. Because the N differed in the different fixation conditions, two analyses were performed; one for the central and 4° eccentric fixation anchors using data from all eight subjects, and a second for all four fixation conditions using the data from the five subjects run in all conditions. SRTs less than 100 ms were regarded as probable anticipations and SRTs greater than 500 ms were regarded as failures to respond. These were eliminated from the data set, as were trials with an initial saccade in the wrong direction or a blink or tracker loss in the interval from 200 ms prior to the target's appearance to the saccade to the target.

In the analysis of the central and 4° fixation anchor data, a total of 85 out of 4608 trials (1.8%) were eliminated, 30 (0.65%) due to short SRTs. We checked the consequence of including these 30 trials in the data, and found this had no effect on the outcomes. There was a main effect of gap condition ($P < 0.001$) but no main effect of fixation anchor type. However, fixation anchor type interacted strongly with gap condition ($P < 0.001$). This interaction is accounted for by a dependence of the 0-Gap to Overlap difference on fixation anchor type. Differences between condition means were tested with two-tailed repeated measures t-tests. With a central fixation point, the data demonstrate a normal two step gap effect, with the mean SRT in the Gap condition 13.9 ms shorter than the mean SRT in the 0-Gap condition ($P < 0.001$), which is 18.2 ms shorter than the mean SRT in the Overlap condition ($P < 0.001$). With the peripheral fixation anchor, the SRT in the Gap condition is 20.3 ms shorter than the SRT in the 0-Gap condition ($P < 0.001$), but the SRT in the 0-Gap condition is not shorter than the SRT in the Overlap condition; it is, in fact, 2.4 ms longer (NS). The loss of a SRT reduction in the 0-Gap condition relative to the Overlap condition with the 4° eccentric fixation anchor was observed in every subject (see Table 1).

In the five subject ANOVA which included all fixation conditions, 126 trials out of 5760 (2.18%) were flagged as errors and eliminated. Fifty of these trials (0.87%) were anticipation errors (SRT < 100 ms). As in the previous analysis, the ANOVA confirmed the presence of a significant main effect for gap condition ($P < 0.001$), absence of a main effect for fixation condition, and significant gap by fixation interaction ($P < 0.001$). As one would expect, condition differences for the central and 4° fixation anchors conditions resemble those obtained with the full group of eight subjects. With the central fixation point, the mean Gap SRT is 15.1 ms shorter than the 0-Gap SRT ($P < 0.001$), and the 0-Gap SRT is 17.7 ms shorter than the Overlap SRT ($P < 0.01$). The corresponding difference values with the 4° fixation anchor are 22 ms ($P < 0.01$) and -2.3 ms (NS). With the 1° fixation anchor gap effects are similar to those found with the central point, with a mean Gap SRT 18.2 ms shorter than the 0-Gap SRT ($P < 0.02$) and the 0-Gap SRT 13.5 ms shorter than the Overlap SRT ($P < 1$[1]). On the other hand, with the 2°

[1] One subject failed to show an RT increase in the Overlap relative to 0-Gap condition with the 1° eccentric fixation anchor (see Table 1). This prevented the difference between the condition means from reaching conventional two-tailed significance. For the four other subjects, the 0-Gap advantage with the 1° fixation anchor resembles that found with a central fixation point.

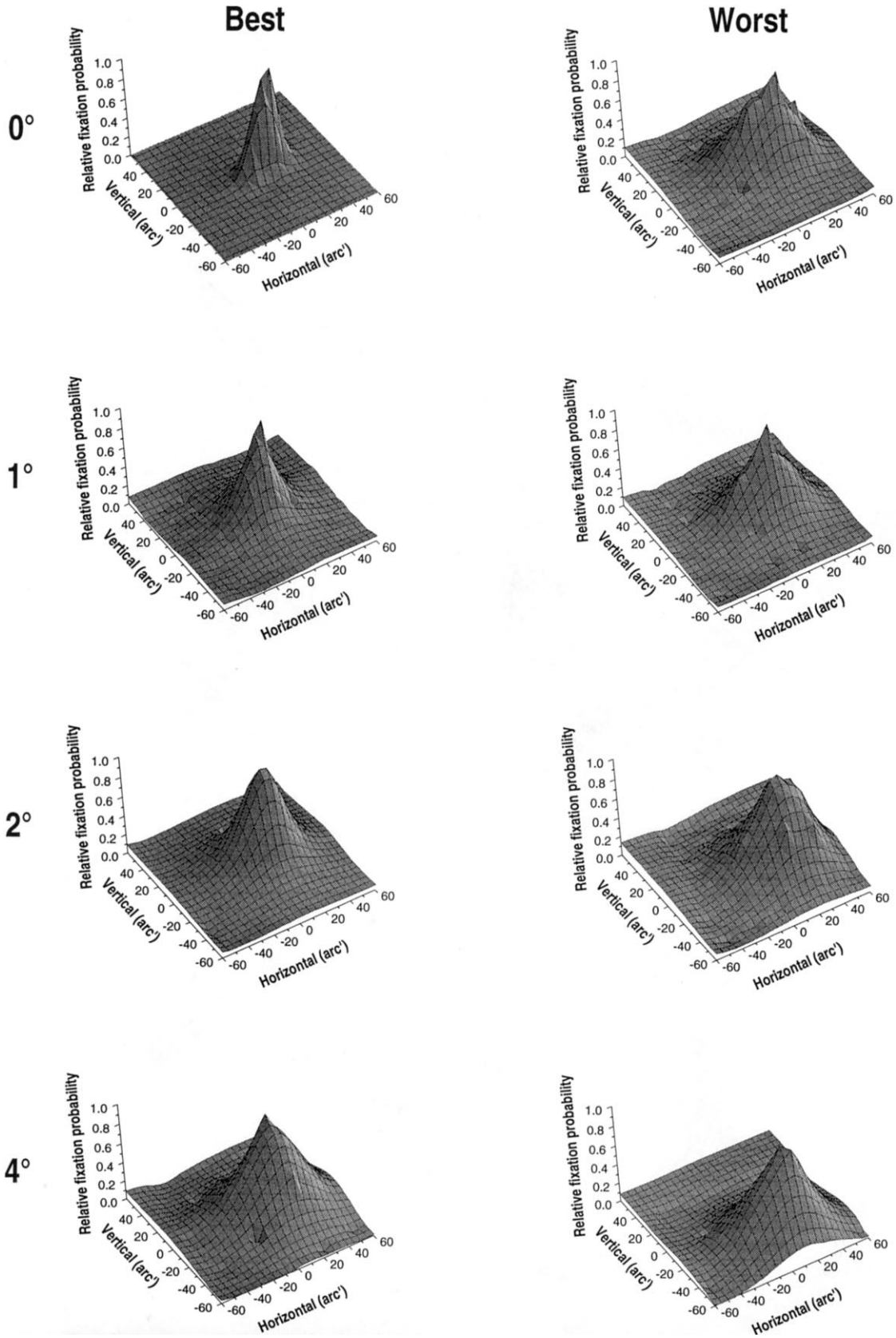

Fig. 2. The distribution of fixation deviations from designated center at the time of the target's appearance with the central and eccentric fixation anchors. Plots are given for the subjects with the best and worst overall fixation accuracy. Axes are in arc min. Plots are bivariate normal surface fits to data generated using 5 arc min bins.

Table 1
Subject and condition mean SRTs for all conditions

Subject	Fixation anchor eccentricity											
	0°			1°			2°			4°		
	Gap 200	Gap 0	Overlap	Gap 200	Gap 0	Overlap	Gap 200	Gap 0	Overlap	Gap 200	Gap 0	Overlap
1	188.6	202.2	215.7	176.0	189.2	187.7	180.6	188.9	190.2	187.3	201.8	201.4
2	227.9	238.4	270.9	213.7	219.6	256.3	222.5	247.0	242.8	219.5	246.9	243.7
3	210.6	223.9	237.7	197.0	215.1	222.9	203.0	213.7	214.9	222.1	235.8	231.3
4	160.0	180.8	199.0	146.3	178.0	191.5	149.8	174.8	177.3	164.0	196.0	192.7
5	170.3	187.6	198.6	173.7	195.8	207.0	179.1	194.7	198.4	164.9	187.3	187.3
6	197.8	209.2	226.5							199.4	212.5	211.1
7	164.2	171.8	184.3							170.3	190.2	179.8
8	192.8	209.2	236.8							219.3	239.2	243.5
Mean 1–5	191.5 (28.0)	206.6 (24.3)	224.3 (30.6)	181.3 (25.5)	199.6 (17.5)	213.1 (27.9)	187.0 (27.4)	203.8 (27.9)	204.7 (25.3)	191.6 (28.3)	213.6 (26.2)	211.3 (24.9)
Mean 1–8	189.0 (23.5)	202.9 (22.3)	221.1 (27.8)							193.4 (25.2)	213.7 (23.7)	211.3 (25.4)

Subject and condition mean reaction times for all conditions. Means designated 1–5 are for the five subjects run in all conditions; means designated 1–8 are for the eight subjects run with the central and 4° eccentric fixation anchors. Data are in ms. Parenthesized values are condition standard deviations.

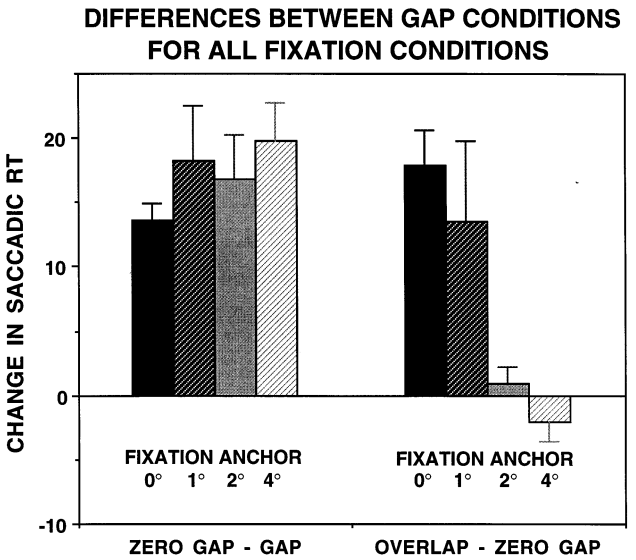

Fig. 3. Differences in ms between the Gap conditions for each fixation condition. Values plotted are the group means for the five subjects run in all conditions. Error bars show standard errors.

fixation anchor the Gap SRT is 16.8 ms shorter than the 0-Gap SRT ($P < 0.01$), but the 0-Gap SRT is only 0.9 ms shorter than the Overlap SRT (NS). The data for the 2° fixation anchor condition therefore resembles that obtained with the 4° fixation anchor: there is no SRT advantage in the 0-Gap condition relative to the Overlap condition. Differences between the gap condition means for the five subjects run in all conditions are graphed in Fig. 3.

3.4. Supplementary analysis

Although the peripheral fixation markers generally allowed subjects to achieve good fixation accuracy, in the trials in which the fixation anchor points were only 1° eccentric, even small fixation errors could have brought the corner points of the square very close to the central fovea. We therefore performed a supplementary analysis of the five subjects run in all conditions in which we considered only trials in which the eye was within 30 arc min of the optimal center position. Averaging across all subjects in each condition, the mean percentage of trials deleted was respectively, 7.7, 6.5, 21.5 and 39.4% for the 0, 1, 2 and 4° fixation anchors. Trimming the data set in this manner produced no change in the pattern of outcomes: SRTs in the Overlap condition were longer than in the 0-Gap condition with the central and 1° eccentric fixations anchors, but not with the 2 and 4° eccentric fixation anchors. Mean condition SRTs for the trimmed data set are given in Table 2. All condition differences that are significant with the full five subject data set remain significant in the supplementary analysis.

4. Discussion

We observed a reduction in SRTs in the Gap condition relative to the 0-Gap condition irrespective of whether the fixation anchor was foveal or eccentric. However, a significant reduction in SRTs in the 0-Gap condition relative to the Overlap condition was observed only when there was a foveal fixation point or the corners of the square forming the eccentric fixation anchor were only 1° from the fovea. When subjects fixated the center of a square formed by four points with an eccentricity of 2 or 4°, mean SRTs in the 0-Gap and Overlap conditions were virtually identical. This absence of an SRT advantage in the 0-Gap condition relative to the Overlap condition was observed in every subject. Fixation instabilities do not appear to account for these results.

Tam & Ono (1994) also investigated the gap effect with eccentric fixation stimuli. When subjects fixated midway between two light emitting diodes separated by 6°, turning off these diodes in a 200 ms gap condition produced a significant SRT facilitation relative to an Overlap condition. This outcome led these investigators to conclude that 'the events leading to the execution of a saccade are similar whether the initially fixated stimulus is foveal or extrafoveal.' However, although they reached a different conclusion, Tam and Ono's data are not incompatible with ours. These investigators also report a smaller Gap to Overlap difference when there was no central fixation point (52 vs 81 ms). Because they did not use a 0-Gap condition, Tam and Ono were not able to observe the essential difference between their foveal and eccentric fixation conditions.

It has been argued that the gap effect in the monkey can be attributed to a more rapid release from active fixation due to the facilitated shutdown of fixation related neurons in the rostral pole of the SC (Munoz & Wurtz, 1992; Dorris & Munoz, 1995). These neurons discharge during active fixation of a foveal target and are suppressed during saccades (Munoz & Wurtz,

Table 2

Fixation anchor eccentricity (°)	Gap condition		
	Gap	0-Gap	Overlap
0	190.9 (28.2)	205.8 (24.4)	223.1 (31.6)
1	181.7 (25.6)	199.1 (17.3)	212.9 (27.3)
2	189.2 (27.8)	205.2 (28.4)	206.3 (24.6)
4	191.2 (23.9)	213.6 (24.5)	211.9 (19.9)

Mean saccadic reaction times for all conditions in the supplementary analysis. Only trials in which a subject's gaze was directed less than 30 arc min from designated center at the time the target appeared were processed. Data are mean reaction times in ms. Parenthesized values are condition standard deviations.

1993b). The disappearance of a central fixation point causes the discharge rate of monkey fixation cells to drop to about 65% of their initial rate after 200–300 ms. If the gap is extended so that the monkey continues to fixate an empty point in space, the discharge rate of the cells increases again but only to about 75% of the initial rate (Dorris & Munoz, 1995).

The present outcomes support the hypothesis that the gap effect in humans measured between the 0-Gap and Overlap conditions can likewise be attributed to the facilitated shutdown of colliculus rostral pole fixation neurons. We propose that in the present experiments, when the fixation anchors were outside the foveal region the retinal signals capable of driving these cells were eliminated. We speculate that in the absence of a retinal input, the activity of these neurons was reduced to a baseline level characteristic of the end of a prolonged gap, allowing their final presaccadic shutdown to be accomplished more rapidly. This would have been equally the case in both the 0-Gap and Overlap conditions. Thus, the 0-Gap and Overlap conditions were rendered equivalent with respect to these cells, so that the disappearance of the eccentric fixation anchors in the 0-Gap condition conferred no relative advantage.

If this interpretation is correct, the data provide psychophysical evidence that in humans the retinal region which projects to fixation cells in the SC extends out by more than 1, but not by 2° from the foveal center. This outcome appears commensurate with physiological data on the distribution of fixation cell receptive fields in the monkey (Munoz & Wurtz, 1993a, 1995b). However, although in the monkey injections of muscimol into the SC rostral pole reduce fixation stability (Munoz & Wurtz, 1993b), it is evident from our own and previous data that humans can maintain fixation using extrafoveal anchors. Gandhi & Keller (1995) and Walker, Kentridge & Findlay (1995) have argued for the presence of fixation related cells in a zone extending out to 10°, and Epelboim & Kowler (1992) have argued that the visual system can maintain fixation by monitoring and correcting for the velocity signals produced by drifts of the eye. The present data do not contradict these hypotheses, but do suggest that special fixation processes operate within 2° of the central fovea. Rattle (1968) has also proposed that at the borders of the fovea there is a shift from one fixation mechanism to another.

In the present data, in contrast to the difference between the 0-Gap and Overlap conditions, the difference between the 200 ms and 0-Gap conditions was unaffected by the nature of the fixation anchor. This outcome suggests that the processes responsible for these gap components are to some degree independent. Other investigators have argued that more than one process can contribute to the gap effect (Fischer & Weber, 1993; Kingstone & Klein, 1993b; Tam & Stelmach, 1993; Paré & Munoz, 1996; Nozawa, Reuter-Lorenz & Hughes, 1995). In addition to the control of fixation by the colliculus rostral pole, it has been proposed that the gap effect may involve the early activation of general motor programming processes (Kingstone & Klein, 1993a; Reuter-Lorenz, Oonk & Barnes, 1995; Bekkering, Pratt & Abrams, 1996). Kingstone & Klein (1993a), for instance, have specifically proposed that the gap effect is driven by two independent processes: a nonspecific motor preparation component and a specifically oculomotor component which they term the 'fixation offset effect.' Alternatively, motor preparatory processes specific to the saccadic system could be occurring in the gap interval (Kowler, 1990; Reuter-Lorenz, Hughes & Fendrich, 1991; Paré & Munoz, 1996; Dorris, Paré & Munoz, 1997). In support of this two-factor hypothesis, Dorris, Paré & Munoz (1997) have recently reported that, in the monkey, variations in saccadic latency within a gap condition can be predicted from the activity level of 'buildup' neurons in the colliculus (Munoz & Wurtz, 1995a) but not the activity level of fixation cells. Another possibility is a process related to the early disengagement of attention (Fischer & Breitmeyer, 1987; Fischer & Weber, 1993), although some investigations have yielded data that appear inconsistent with this idea (Kingstone & Klein, 1993a; Walker, Kentridge & Findlay, 1995).

If one assumes that two processes are contributing to the gap effect—fixation release mediated by the SC rostral pole and a saccade preparatory process which does not require a foveal stimulus but does require a non-zero gap interval—then the outcomes of the present experiments can be explained as follows. We presume that some processes involved in the initiation of saccades can be implemented prior to fixation release, while other processes can only be completed after fixation release has been achieved. Kowler (1990) has previously discussed the viability of early partial saccadic preprogramming:

1. with a foveal fixation anchor, in the Gap condition the SRT advantage relative to the Overlap condition is produced by both advance saccade motor preparations and facilitated fixation release. These components act in combination, yielding a maximum advantage in the Gap condition.
2. With a foveal fixation anchor, in the 0-Gap condition saccade preparation begins at the same time as in the Overlap condition. However, in the absence of a fixation point, fixation release can be accomplished more rapidly in the 0-Gap condition. Since only more efficient fixation release contributes to the gap effect, the observed SRT facilitation is smaller than in (1).
3. With an eccentric fixation anchor, in the Gap condition both advance saccadic preparations and a facilitated fixation release act to reduce SRTs. However,

fixation release is also facilitated in the Overlap condition. Relative to the Overlap condition, the SRT advantage in the Gap condition is therefore smaller than in (1).

4. With an eccentric fixation anchor, fixation release occurs with equal facility in the 0-Gap and Overlap conditions, and saccadic programming begins at the same time in these two conditions. Therefore, there is no SRT advantage in the 0-Gap relative to the Overlap condition.

Whether or not this account of our data proves correct, motor preparations which do not depend on fixation release are likely to contribute to the gap effect. In the present experiments, if a warning tone had not been used a general warning produced by the offset of the fixation point might well have played an increased role in the data (Reuter-Lorenz, Oonk & Barnes, 1995). Likewise, when the location of the saccadic target is predictable, preparatory motor programs probably play an increased role in the reduction of saccadic reaction times (Fischer & Ramsperger, 1986; Paré & Munoz, 1996) in both monkey and man. Bimodal SRT distributions with distinct populations of very short latency 'express saccades' are sometimes found in gap paradigms (Fischer & Boch, 1983; Fischer & Ramsperger, 1984; Fischer, Weber, Biscalkdi, Aiple, Otto & Stuhr, 1993; Schiller, Sandell & Maunsell, 1987; McPeek & Schiller, 1994; Paré & Munoz, 1996), although in the present experiment we observed no evident cases of bimodality when we inspected the latency distributions of our subjects. The occurrence of a distinct population of express saccades may depend on an interaction of several facilitatory factors. Nozawa, Reuter-Lorenz & Hughes (1995) have generated a stochastic model showing how this might occur, and Dorris, Paré & Munoz (1997) have suggested that express saccades are contingent upon the direct modulation of buildup neurons in the SC, possibly by descending projections from the frontal eye fields (Seagraves & Goldberg, 1987; also, see Dias & Bruce, 1994). A full account of the gap effect will therefore almost certainly involve multiple processes. However, the present data indicate that one component of this effect—the SRT difference found between the 0-Gap and Overlap conditions—occurs only when the fixation anchor is foveal. This component may therefore be mediated by a single neural module in the colliculus rostral pole.

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